Original Article

MEDICINE PALLIATIVE CARE

DOI: 10.47582/jompac.1592028

J Med Palliat Care. 2025;6(1):20-26

Prolonged atrial electromechanical delay and P-wave parameters in asymptomatic carotid artery stenosis: novel insights from a non-invasive evaluation

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Cite this article as: Serhatlıoğlu F, Keleşoğlu Ş. Prolonged atrial electromechanical delay and P-wave parameters in asymptomatic carotid artery stenosis: novel insights from a non-invasive evaluation. *J Med Palliat Care*. 2025;6(1):20-26.

Received: 27.11.2024 ◆ **Accepted**: 13.01.2025 ◆ **Published**: 14.02.2025

ABSTRACT

Aims: Stroke is one of the top three causes of death in developed societies. Ischemic strokes are linked to carotid artery stenosis (CAS). Atrial fibrillation (AF) is a commonly encountered clinical arrhythmia. It has been shown that the prolongation of intra- and interatrial conduction times, known as atrial electromechanical delay (EMD), is associated with a higher risk of AF. We aimed to determine the correlation of atrial conduction abnormalities between surface electrocardiographic and TDI measurements in the CAS patient group.

Methods: The study included 76 patients diagnosed with extracranial internal carotid artery (ICA) stenosis. Asymptomatic severe CAS was defined as patients with 70-99% stenosis detected by carotid digital subtraction angiography (DSA). The longest P-wave and the longest atrial conduction time ACT were considered as the maximal P-wave duration. The difference between the longest P-wave (Pmax) and the shortest P-wave (Pmin) was accepted as PD. (PD=Pmax-Pmin). Atrial EMD was defined as the time interval from the onset of atrial electrical activity to the beginning of mechanical atrial contraction.

Results: The CAS group had significantly longer Pmax and PD values compared to the control group (Pmax 104.72±6.03 and 93.06±7.26 ms, p<0.001; PD 48.55±6.72 and 38.50±8.12 ms, p<0.001). In the TDI examination, the atrial EMD parameters (PA lateral, PA septum) were significantly longer in the CAS group compared to the control group. (77.88±5.13 vs 65.53±9.11 ms; p<0.0001; 63.77±3.95 vs 54.56±7.13 ms; p<0.001 respectively) Both interatrial and intra-atrial EMD times were found to be longer in the CAS group compared to the control group (31.72±7.39 vs 22.13±8.67 ms; p<0.001; 17.61±7.76 vs 11.16±7.76 vs 11.16±7.04 ms; p<0.001, respectively). In the correlation analysis, a positive relationship was found between interatrial and interatrial EMD and Pmax and PD (p<0.001, both).

Conclusion: We found that both intra-atrial and inter-atrial electromechanical conduction times were longer in CAS patients. This suggests that CAS patients are at risk for AF in their follow-up.

Keywords: Carotid artery stenosis, atrial fibrillation, atrial conduction time

INTRODUCTION

Stroke is one of the top three causes of death in developed societies. About 80% of strokes have an ischemic origin, and 15-20% of ischemic strokes are linked to carotid artery stenosis (CAS).[1,2] Atherosclerotic stenosis of the internal carotid artery (ICA) is found in 1-2% of adults in the general population, although >10% of those between the ages of 60 and 79 are affected.[3,4]

Atrial fibrillation (AF) is a commonly encountered clinical arrhythmia that causes hemodynamic disturbances, frequent hospitalizations, and thromboembolic events, affecting 1-2% of the general population.[5] Although the exact mechanisms causing AF are not fully understood, atherosclerotic risk factors such as hypertension (HT), diabetes mellitus (DM), advanced age, endothelial dysfunction, and increased oxidative stress play significant roles in the pathogenesis of AF.[6] As is known, there are similar risk factors in the pathogenesis of coronary artery disease (CAD), just like in AF.[7] Therefore, the risk of developing new AF may increase in these patients.

Atrial conduction time (ACT) represents the interval between sinus impulses and atrial mechanical contraction. As an alternative to invasive electrophysiological measurements, it can be analyzed non-invasively by measuring with tissue Doppler echocardiography imaging (TDI).[8] It has been shown that the prolongation of intraatrial and interatrial conduction times, known as atrial electromechanical delay (EMD), is associated with a higher risk of AF.[9,10] At the same time, it has been shown that P-wave dispersion (PD)

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and maximum P-wave (Pmax) duration can be noninvasive electrocardiographic indicators of AF.[10,11]

To our knowledge, there is no study evaluating atrial conduction abnormalities in patients with CAD using non-invasive tests such as TDI and ECG. In this study, we aimed to determine the correlation of atrial conduction abnormalities between surface electrocardiographic and TDI measurements in the CAS patient group.

METHODS

The study was conducted with the permission of Erciyes University Faculty of Medicine Clinical Researches Ethics Committee (Date: 24.11.2021, Decision No: 2021/767). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

The retrospective study included 217 patients diagnosed with extracranial ICA stenosis at our institution between March 2022 and July 2023. Patients with symptomatic CAS, a history of ischemic or hemorrhagic stroke, a history of CAD, segmental or global wall motion abnormalities, evidence of moderate to severe heart valve disease on echocardiography, structural heart disease, permanent or paroxysmal AF, bundle branch block or any conduction disorder on ECG, those who underwent pacemaker implantation, patients with known thyroid disease and chronic obstructive pulmonary disease, and those with electrolyte imbalances that could affect atrial EMD were excluded from the study. Asymptomatic individuals with risk factors such as HT, diabetes, or a family history of cardiovascular disease, who underwent carotid artery ultrasound for the purpose of subclinical disease screening, and in whom critical stenosis (>70%) was detected in either the right or left carotid artery, were included. Patients with risk factors but normal carotid Doppler findings formed the control group. We studied patients with 70% or more carotid stenosis on Doppler ultrasonography (DUSG) in the study for CAAG. So patients with asymptomatic severe CAD were included in the study. 141 patients who did not meet the inclusion criteria were excluded from the study. Finally, 76 patients were analyzed. Asymptomatic severe CAS was defined as patients with 70-99% stenosis detected by carotid digital subtraction angiography (DSA) in the right and/or left extracranial ICA without a transient ischemic attack or stroke in the past 6 months. According to current guidelines for CAS, patients were started on medical treatment and underwent revascularization therapy. Before the planned treatments, all patients underwent detailed transthoracic echocardiographic examinations.

Detailed medical history, physical examination, 12-lead electrocardiography (ECG), complete blood count, and serum biochemistry were obtained from all patients. The presence of classical cardiovascular risk factors such as HT, DM, and hyperlipidemia was evaluated. DM, HT, and hyperlipidemia were defined as previously described.[12]

Electrocardiography

ECG recordings were performed with at least 3 QRS complexes for each derivation, at a speed of 25 mm/sec, with an amplitude of 1 mV, and in standard 12 derivations using a 3-channel simultaneous Philips brand machine ECG device. During the recording, patients were allowed to breathe comfortably, but they were not permitted to speak. The P-wave durations in all derivations were manually measured using calipers and magnifying lenses to reduce measurement errors.

The beginning of the P-wave was taken as the point where the isoelectric line intersects with the P-wave. The endpoint was taken as the intersection of the isoelectric line and the endpoint of the P-wave. The longest P-wave and the longest ACT were considered as the maximal P-wave duration. The difference between the longest P-wave (Pmax) and the shortest P-wave (Pmin) was accepted as PD (PD=Pmax-Pmin). All calculations were evaluated separately in a single-blind manner by two cardiology specialists who were unaware of the patients' clinical characteristics, and the average of these two values was accepted as PD and maximum P-wave duration.

Echocardiography

Conventional echocardiography was performed with 2-dimensional, M-mode, pulsed wave, continuous, color Doppler and tissue Doppler imaging using Philips Epiq 7 ultrasound system (Philips, Andover, Mass., USA). Simultaneous ECG recording was done. All patients were in sinus rhythm at the time of examination. Conventional echocardiographic images were obtained from the parasternal and apical views according to the guidelines of the American Society of Echocardiography.[14] Left ventricular (LV) diameters and wall thickness were measured from the para- sternal views by M-mode echocardiography. The Teichholz method was used for the calculation of LV ejection fraction. The left atrial area and diameter were measured from the parasternal long axis view. Mitral inflow velocities were measured from apical views.

Atrial Electromechanical Time Measurement

TDI was performed using transducer frequencies of 3.5–4.0 MHz. The spectral pulsed Doppler signal filters were adjusted until a Nyquist limit of 15–20 cm/s was obtained. The minimal opti- mal gain was used. Myocardial TDI velocities [peak systolic (S'), early diastolic (E') and late diastolic velocities (A')] were measured with spectral pulsed Doppler from the apical 4-chamber view. The ultrasound beam slope did not exceed 15% to acquire the optimal angle of imaging. The monitor sweep speed was adjusted at 50–100 mm/s to optimize the spectral display of myocardial velocities. Atrial EMD was defined as the time interval from the onset of atrial electrical activity (P-wave on surface ECG) to the beginning of mechanical atrial contraction (late diastolic A-wave). All values were averaged over 3 consecutive beats. Atrial EMD was measured from the lateral mitral annulus and called 'PA lateral', from the septal mitral annulus, called 'PA septal', and from the right ventricle tricuspid annulus, called 'PA tricuspid'. Interatrial EMD was calculated as the difference between PA lateral and PA tricuspid, intra-atrial EMD was calculated as the difference between PA septum and PA tricuspid, and left-atrial EMD was calculated as the difference between PA lateral and PA septum.[8]

Statistical Analysis

Statistical analyzes were performed using SPSS Statistics Package version 21.0 (SPSS Inc, Chicago, IL, USA) for Windows. The distribution characteristics of the data were determined by using Kolmogorov–Smirnov test. Independent Sample t test was used for Parametric scale variables. Mann–Whitney U test was used for nonparametric scale variables. The $\chi 2$ test was used for univariate analysis of the categorical variables. The variables were given as means±SD; categorical variables were defined as percentages. Correlation analyses were per- formed using Pearson's coefficient of correlation and Spearman coefficient of correlation. A probability value of $p<0.05$ was considered significant, and 2-tailed p values were used for all statistics.

RESULTS

The baseline characteristics of the patients and the control group are presented in Table 1. The average ages of the CAS group and the control group were 63.4±6.4 and 62.5±7.4, respectively. There was no significant difference between the patients and the control group in terms of demographic parameters such as age, gender, HT, DM, and smoking. Among the baseline blood parameters, the low-density lipoprotein cholesterol (LDL-C) level was significantly higher compared to the control group, while the high-density lipoprotein cholesterol (HDL-C) level was significantly lower (p=0.048, p=0.026, respectively). Other blood parameters were similar between the groups.

The CAS group had significantly longer Pmax and PD values compared to the control group. (Pmax 104.72±6.03 and 93.06±7.26 ms, p<0.001; PD 48.55±6.72 and 38.50±8.12 ms, p<0.001) However, the Pmin values did not show a significant difference (Table 2).

Echocardiographic and atrial electromechanical time parameters are shown in Table 3. LV systolic and diastolic diameters, interventricular septum and LV posterior wall thickness, and LV ejection fraction were similar in both groups. No significant difference was observed between the groups in the left atrium diameters and the parameters indicating LV diastolic functions, namely isovolumetric relaxation time and deceleration time.

In the TDI examination, the atrial EMD parameters (PA lateral, PA septum) were significantly longer in the CAS group compared to the control group. (77.88±5.13 vs 65.53±9.11 ms; p<0.001; 63.77±3.95 vs 54.56±7.13 ms; p<0.001 respectively). The tricuspid PA, on the other hand, was similar in both groups (46.16±6.08 vs 43.40±6.96 ms: p=0.090).

Both interatrial (PA lateral–PA tricuspid) and intraatrial (PA septal–PA tricuspid) EMD times were found to be longer in the CAS group compared to the control group (31.72±7.39 vs 22.13±8.67 ms; p<0.001; 17.61±7.76 vs 11.16±7.76 vs 11.16±7.04 ms; p<0.001, respectively). Left atrial EMD (PA lateral-PA septal) was similar between the two groups. (14.11±6.58 vs 10.96±8.60 ms: p=0.098) (Figure 1).

In the correlation analysis, a positive relationship was found between interatrial EMD and Pmax and PD (r=0.617, p<0.05 and r=0.308, p<0.05, respectively). Similarly, a similar relationship was observed between intra-atrial EMD and Pmax and PD (r=0.333, p<0.05 and r=0.372, p<0.001, respectively) (Figure 2).

DISCUSSION

In our study, three important findings identified in patients with CAS can be listed as follows:

Table 1. Baseline clinical and demographic features of the study groups

Variables	Control group (n=54)	Carotis artery disease (n=76)	p-value
Age (years)	62.56±7.48	63.44±6.40	0.609
Male/female	17/13	21/15	0.891
HT	11 (36.6%)	17 (47.2%)	0.388
DM	7 (23.3%)	11 (30.55%)	0.512
Smoke	7 (23.3%)	11 (30.55%)	0.512
Systolic blood pressure, mm Hg	120.23±12.30	119.13±10.14	0.693
Diastolic blood pressure, mm Hg	73.16±6.42	74.47±7.48	0.455
Glucose (mg/dl)	93.70±14.10	101.94±24.35	0.106
Creatinine (mg/dl)	0.84±0.20	0.95±0.27	0.059
Total cholesterol (mg/dl)	197.38±48.49	204.05±36.65	0.527
HDL cholesterol (mg/dl)	47.35±8.32	42.22±9.70	0.026*
LDL cholesterol (mg/dl)	115.70±39.19	132.83±29.64	0.048*
TG (mg/dl)	168.05±86.81	171.05±71.37	0.878
AST (U/L)	18.83±6.13	22.47±13.39	0.175
ALT (U/L)	18.36±7.69	18.83±13.80	0.870
WBC (10^3/uL)	7.69±1.49	7.75±2.20	0.904
Hemoglobin (g/dl)	13.90±1.67	17.80±20.97	0.315
Platelet (x10^3/mm^3)	259.26±65.19	258.68±76.05	0.974

Data are expressed as mean±standard deviation for normally distributed data and percentage (%) for categorical variables. DM: Diabetes mellitus, HT: Hypertension, WBC: White blood cell, HDL: High-density lipoprotein, LDL: Low-density lipoprotein

Table 2. Electrocardiographic characteristics of the study population

Variables	Control group (n=54)	Carotis artery disease (n=76)	p-value
Heart rate (min)	77.33±10.90	80.05±2.31	0.149
Pmax (ms)	93.06±7.26	104.72±6.03	p<0.01
Pmin (ms)	54.56±3.53	56.16±3.67	0.077
PD (ms)	38.50±8.12	48.55±6.72	p<0.01

Pmax: Maximum P-wave duration, Pmin: Minimum P-wave duration, PD: P-wave dispersion, Min: Minute, ms: Millisecond

Table 3. Echocardiography characteristics of the study population

Variables	Control group (n=54)	Carotis artery disease (n=76)	p-value
LA Diameter, cm	3.36±0.32	3.50±0.26	0.057
LVEDD, cm	4.72±0.32	4.75±0.45	0.747
LVESD, cm	3.00±0.35	3.06±0.32	0.458
IVSD, cm	1.06±0.11	1.07±0.16	0.847
PWD, cm	1.04±0.08	1.07±0.15	0.370
LVEF, %	66.96±4.44	64.69±4.74	0.051
PA lateral, ms	65.53±9.11	77.88±5.13	**p<0.01**
PA septum, ms	54.56±7.13	63.77±3.95	**p<0.01**
PA tricuspid, ms	43.40±6.96	46.16±6.08	0.090
PA lateral-PA tricuspid (Inter-atrial delay)	22.13±8.67	31.72±7.39	**p<0.01**
PA septal-PA tricuspid (Intra-atrial delay)	11.16±7.04	17.61±7.76	**p<0.01**
PA lateral- PA septal (Left-atrial delay)	10.96±8.60	14.11±6.58	0.098
Mitral E, cm/S	7.6±1.17	7.30±1.25	0.328
Mitral A, cm/S	5.95±1.61	6.27±1	0.324
DT, ms	170.76±23.04	165.61±29.8	0.442
IVRT, ms	86.7±10.33	89.13±8.83	0.305
(S') cm/s	11±3.26	11.02±2.47	0.969
(E') cm/s	13.66±3.4	12.52±2.64	0.131
(A') cm/s	9.46±2.6	10.94±2.26	0.017*

LA: Left atrium, LVEDD: LV end-diastolic dimension, LVESD: LV end-systolic dimension, IVSD: Interventricular septum thickness, PWD: Posterior wall thickness, LVEF: LV ejection fraction, DT: Deceleration time, IVRT: Isovolumic relaxation time, Interatrial delay: PA lateral-PA tricuspid, Intra-atrial delay: PA septum-PA tricuspid, Left-atrial delay: PA lateral-PA septum, S': Systolic velocity from the mitral annulus, E': Early diastolic velocity from the mitral annulus, A': Late diastolic velocity from the mitral annulus

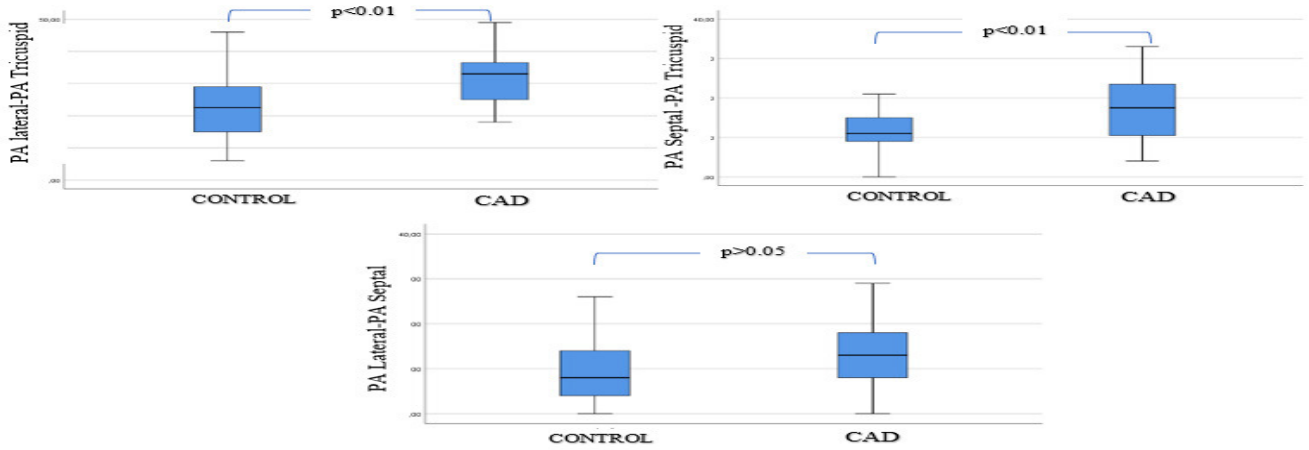

Figure 1. Change PA lateral-PA tricuspid, PA septal-PA tricuspid, PA lateral-PA septal between study groups

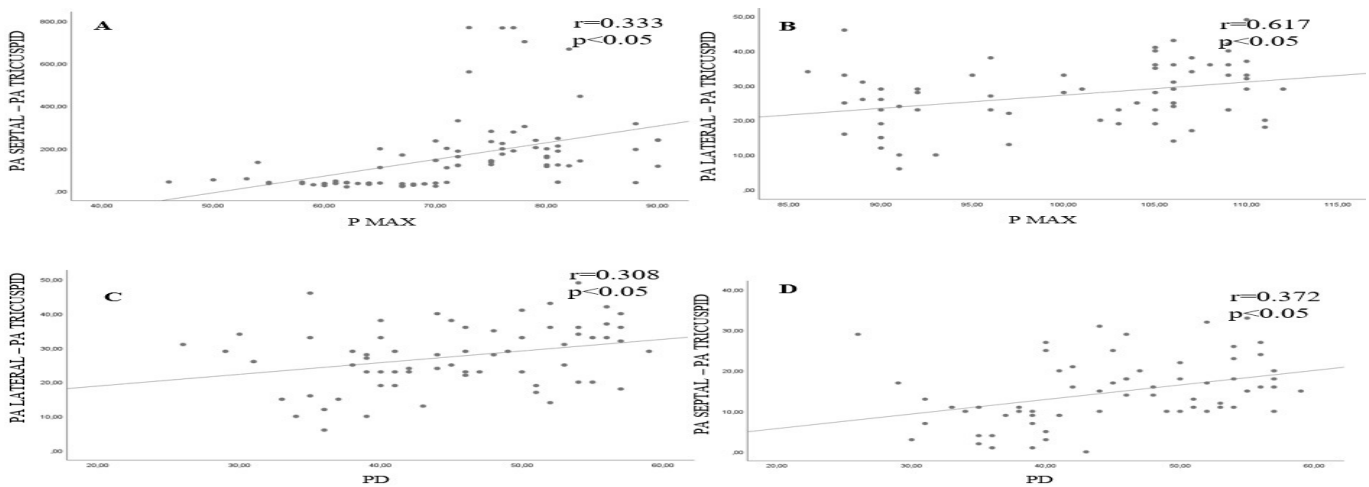

Figure 2. Correlation between PA septal-PA tricuspid and Pmax duration. (**A**) Correlation between PA lateral-PA tricuspid and Pmax duration. (**B**) Correlation between PA lateral-PA tricuspid and PD duration. (**C**) Correlation between PA septal-PA tricuspid and PD duration (**D**)
Pmax: Maximum P-wave duration, PD: P-wave dispersion

(1) The PD and Pmax durations being longer in the 12-lead surface ECG compared to the control group; (2) The duration of both intraatrial and interatrial electromechanical conduction being longer as detected by TDI; (3) The PD and Pmax durations showing a significant correlation with the intraatrial and interatrial electromechanical coupling durations.

AF is the most common arrhythmia in the community, causing an increase in cardiovascular mortality and morbidity.[15] AF is important to recognize early because it leads to stroke and thromboembolism in patients, increasing the risk of mortality and morbidity, thereby reducing the quality of life. In addition to all these effects, it also imposes a significant financial burden on society. Hospital stays, procedure and medication costs, along with absenteeism costs, lead to significant expenses.[16] Therefore, it is important to determine which patients with CAS are at higher risk for developing AF.

When we evaluate the literature, there are conflicting results regarding the prevalence of AF in patients with CAS. As is known, atherosclerotic vascular diseases are an important risk factor for the development of AF and are often found together. Adamsson Eryd et al.[17] in their study, demonstrated that both carotid atherosclerosis and high carotid artery intima-media thickness (IMT) are associated with an increased risk of AF occurrence over an average follow-up period of 15 years. They even claimed that carotid IMT showed a similar effect to HT and heart failure, which are among the strongest risk determinants for AF development. Willeit et al.[18] in their study, found a higher risk of AF development in individuals with carotid atherosclerosis compared to those without. Supporting all these studies, the Rotterdam study, although differing in details, also showed that the presence of carotid atherosclerosis measured ultrasonographically could predict the future risk of AF.[19] Luo et al.[20] claimed that AF was associated with a higher recurrence rate after radiofrequency catheter ablation in patients with carotid atherosclerosis.

In a study conducted by Chen LY et al.[21] on young patients with lone AF, they found a significant association between high carotid IMT and carotid-femoral pulse wave velocity and the development of AF. In addition to Pmax and PD, which can be easily measured with an ECG, the evaluation of Atrial EMD with TDI is one of the precursors to the risk of developing new-onset AF. Therefore, in this study, we aimed to assess the risk of AF in patients with CAS by examining the aforementioned parameters.

Pmax and PD have been used to predict the risk of AF in patients with paroxysmal AF, mitral stenosis, aortic stenosis, dilated cardiomyopathy (DCM), acute myocardial infarction, atherosclerotic heart disease, ischemia with no obstructive arteries, primary hyperparathyroidism and angina.[22-28] Atrial EMD, on the other hand, has been found to be longer in individuals with paroxysmal AF and mitral stenosis compared to controls, and this condition has been reported to be associated with PD.[29] Additionally, it has been shown in previous studies that atrial EMD increases in many clinical disorders such as DM, HT, and non-ischemic DCM.[30-34] In conclusion, both atrial EMD and Pmax and PD have frequently been used as non-invasive markers to predict the risk of AF development.

In our study, the average PD and Pmax values were found to be significantly longer in patients with CAS compared to the control group. Moreover, the intraatrial and interatrial EMD were significantly longer and showed a significant correlation with PD and Pmax durations. Some plausible reasons can be suggested for the increased risk of AF in patients with CAS. One of these is closely related to carotid atherosclerosis and wall thickening, coronary atherosclerosis, and microvascular damage.[35-37] This condition can lead to hypoperfusion and ischemia of the atrium, and subsequently to fibrosis. An increase in atrial fibrosis can cause prolonged intraatrial/interatrial conduction times and non-homogeneous propagation of sinus impulses.[38] On the other hand, with advancing age, the atherosclerotic process can affect the carotid arteries as well as the aorta and other peripheral vessels, leading to an increase in systolic load and aortic stiffening. The changes that occur at the end of this process may lead to ventricular remodeling as well as atrial remodeling, and over time, all these changes in the heart may have laid the groundwork for the prolongation of the aforementioned parameters.[39-41]

It is known that the increase in the left atrial diameter is significant in the development of AF.[42] However, in the study by Tükek et al., it has been reported that in patients with paroxysmal AF, PD is prolonged even when the atrial diameter is normal.[43] In our study, no significant difference was observed between the patient and control groups in terms of left atrial diameter.

Current guidelines recommend echocardiography and 24-72 hour rhythm Holter monitoring in patients with 50-99% ICA stenosis who have recently experienced a transient ischaemic attack or stroke.[44]

CONCLUSION

In our study, we demonstrated that intraatrial and interatrial EMD, Pmax, and PD, which are techniques predicting the risk of future AF development, were significantly longer in asymptomatic patients with CAS compared to controls. Since atrial EMD is increased in paroxysmal AF and is considered a predictor of new-onset AF, patients with severe CAD who have not yet experienced an ischemic stroke should also be investigated for paroxysmal AF, and if necessary, rhythm Holter monitoring should be considered for this patient group.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the permission of Erciyes University Faculty of Medicine Clinical Researches Ethics Committee (Date: 24.11.2021, Decision No: 2021/767).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement
The authors have no conflicts of interest to declare.

Financial Disclosure
The authors declared that this study has received no financial support.

Author Contributions
All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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